Cerebral Venous Sinus Thrombosis Pathogenesis

There are many uncontrollable factors in the pathogenesis of cerebral venous sinus thrombosis (CVST). In order to further explore the pathophysiology and morphology of CVST, it is necessary to establish a highly compatible CVST animal model that can standardize the site and stage of venous thrombosis.

Animal model

A study employed the insertion of a self-made thread embolism into the superior sagittal sinus (SSS) to establish a rat model of SSS occlusion that emulates CVST. The thread embolism was removed after 6 h of SSS occlusion in order to achieve recanalization. After the successful preparation of the model, the cerebral blood flow (CBF) status and ultrastructural changes of the blood brain barrier (BBB) were monitored.

This CVST model was able to achieve continuously high occlusion of SSS. The parasagittal venous-collateral circulation underwent extensive compensation and recombination, which alleviated blood flow stasis and brain tissue hypoxia caused by restricted reflux. Removing SSS occlusion significantly improved cerebral circulation, reduced brain edema, and accelerated the receding of brain edema.

This study established a new model of acute occlusion and recanalization of SSS in rats via a thread-embolism method, which standardized the ischemic site and stage of venous thrombosis. In addition, the study suggests that promoting collateral circulation may be a potential treatment for promoting brain protection [1].

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Wang W, Mu S, Xu W, Liang S, Lin R, Li Z, Liu Q, Yi G, Xue L, Wang S. Establishment of a Rat Model of Superior Sagittal-Sinus Occlusion and Recanalization Via a Thread-Embolism Method. Neuroscience. 2019 Aug 1. pii: S0306-4522(19)30535-4. doi: 10.1016/j.neuroscience.2019.07.044. [Epub ahead of print] PubMed PMID: 31377451.